Psychosocial Stress Over the Lifespan, Psychological Factors, and Cardiometabolic Risk in the Community

Gebreab, Sirak Zenebe ; Vandeleur, Caroline L ; Rudaz, Dominique ; Strippoli, Marie-Pierre F ; Gholam-Rezaee, Mehdi ; Castelao, Enrique ; Lasserre, Aurélie M ; Glaus, Jennifer ; Pistis, Giorgio ; Kuehner, Christine ; von Känel, Roland ; Marques-Vidal, Pedro ; Vollenweider, Peter ; Preisig, Martin

Abstract: OBJECTIVE: The complex relationship between psychosocial stress over the lifetime, psychological factors, and cardiometabolic risk is still poorly understood. Accordingly, our aims were (1) to independently assess the associations between childhood adversity, life-event stress in remote (earlier than the last 5 years), and recent adulthood and cardiometabolic risk, and (2) to determine the role of psychological factors including personality, coping, and depression in these associations. METHODS: The sample included 2674 adults, aged 35 to 66 years, randomly selected from urban area. Participants underwent a physical examination including the assessment of obesity markers, blood pressure, and blood lipid and glucose levels. Stress during adulthood was determined using the severity scores of 52 stressful life events. Information on adverse childhood experiences and major depressive disorders was collected using semistructured interviews, whereas personality traits and coping mechanisms were evaluated through questionnaires. RESULTS: Both childhood adversity and stress in remote adulthood were associated with elevated body mass index ([95% confidence interval CI] = 0.249 [0.029 to 0.468]; 0.020 [0.006 to 0.034]), waist circumference ([95% CI] = 0.061 [0.024 to 0.099]; 0.08 [0.04 to 0.11]), and the global cardiometabolic risk score ([95% CI] = 0.278 [0.017 to 0.540]; 0.017 [0.001 to 0.033]) after adjustment for sociodemographic, lifestyle, and psychological factors. In addition, childhood adversity was associated with low high density lipoprotein levels ([95% CI] = -0.021 [-0.042 to 0.000]), as well as increased fat mass and systolic blood pressure levels ([95% CI] = 0.506 [0.165 to 0.846]; 0.952 [0.165 to 1.740]) and stress in remote adulthood with apolipoprotein B levels ([95% CI] = 0.607 [0.312 to 0.901]). Psychological factors did not account for these associations and were not effect modifiers. CONCLUSIONS: Our data demonstrate that psychosocial stress during childhood and remote adulthood favor adiposity and abnormal lipid metabolism.

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Sirak Zenebe Gebreab, MSc, Caroline L. Vandeleur, PhD, Dominique Rudaz, MSc, Marie-Pierre F. Strippoli, MSc, Mehdi Gholam-Rezaee, PhD, Enrique Castelao, MSc, Aurélie M. Lasserre, MD, PhD, Jennifer Glaus, PhD, Giorgio Pistis, PhD, Christine Kuehner, PhD, Roland von Känel, MD, Pedro Marques-Vidal, MD, PhD, Peter Vollenweider, MD, and Martin Preisig, MD, MPH

ABSTRACT

Objective: The complex relationship between psychosocial stress over the lifetime, psychological factors, and cardiometabolic risk is still poorly understood. Accordingly, our aims were (1) to independently assess the associations between childhood adversity, life-event stress in remote (earlier than the last 5 years), and recent adulthood and cardiometabolic risk, and (2) to determine the role of psychological factors including personality, coping, and depression in these associations.

Methods: The sample included 2674 adults, aged 35 to 66 years, randomly selected from urban area. Participants underwent a physical examination including the assessment of obesity markers, blood pressure, and blood lipid and glucose levels. Stress during adulthood was determined using the severity scores of 52 stressful life events. Information on adverse childhood experiences and major depressive disorders was collected using semistructured interviews, whereas personality traits and coping mechanisms were evaluated through questionnaires.

Results: Both childhood adversity and stress in remote adulthood were associated with elevated body mass index (β [95% confidence interval {CI}] = 0.249 [0.029 to 0.468]; 0.020 [0.006 to 0.034]), waist circumference (β [95% CI] = 0.061 [0.024 to 0.099]; 0.08 [0.04 to 0.11]), and the global cardiometabolic risk score (β [95% CI] = 0.278 [0.017 to 0.540]; 0.017 [0.001 to 0.033]) after adjustment for sociodemographic, lifestyle, and psychological factors. In addition, childhood adversity was associated with low high density lipoprotein levels (β [95% CI] = −0.021 [−0.042 to 0.000]), as well as increased fat mass and systolic blood pressure levels (β [95% CI] = 0.506 [0.165 to 0.846]; 0.952 [0.165 to 1.740]) and stress in remote adulthood with apolipoprotein B levels (β [95% CI] = 0.607 [0.312 to 0.901]). Psychological factors did not account for these associations and were not effect modifiers.

Conclusions: Our data demonstrate that psychosocial stress during childhood and remote adulthood favor adiposity and abnormal lipid metabolism.

Key words: cardiometabolic risk, childhood adversity, life events, major depressive disorder, personality, psychosocial stress.

INTRODUCTION

Epidemiological and clinical evidence has revealed that both cardiovascular diseases (CVDs) and their risk factors are associated with psychosocial stress (1–4). Cardiometabolic abnormalities such as an increase in body weight, especially high fat distribution in the central region, have been linked with chronic stress (5–8) Likewise, high levels of blood glucose and blood lipids have been found to be associated with life stress (4,9,10). Although findings vary, psychosocial stress has also been associated with high blood pressure (11). Research is also increasingly recognizing the role of stress during childhood as a risk factor for cardiometabolic abnormality in adulthood (12–16). Indeed, adverse childhood experiences such as stressful life events, maladaptive family environments, sexual abuse, and physical abuse have been shown to be associated with cardiometabolic risk factors such as increased body weight (17) and diabetes (18,19). Stress during childhood has also been shown to affect overall cardiometabolic functioning later on in life (20,21).

Despite the growing body of evidence in this domain, exposure to psychosocial stressors over the entire life and the risk of cardiometabolic abnormality is only partially elucidated. One major limitation is that researchers often examine stress exposure within limited periods of time, whereas stressors are ubiquitous and span

BMI = body mass index, **CVDs** = cardiovascular diseases, **HDL** = high density lipoprotein, **MDD** = major depressive disorder, **SBP** = systolic blood pressure

SDC Supplemental Content

From the Department of Psychiatry, Center for Psychiatric Epidemiology and Psychopathology (Gebreab, Vandeleur, Rudaz, Strippoli, Gholam-Rezaee, Castelao, Lasserre, Glaus, Pistis, Preisig), and Department of Medicine, Internal Medicine (Marques-Vidal, Vollenweider), Lausanne University Hospital, Switzerland; Genetic Epidemiology Research Branch (Glaus), Intramural Research Program, National Institute of Mental Health, Bethesda, Maryland; Longitudinal and Intervention Research (Kuehner), Central Institute of Mental Health, Medical Faculty Mannheim, Heidelberg University, Mannheim, Germany; and Department of Consultation-Liaison Psychiatry and Psychosomatic Medicine (von Känel), Zürich University Hospital, Switzerland.

Address correspondence to Sirak Zenebe Gebreab, MSc, Lausanne University Hospital, Department of Psychiatry, Center for Psychiatric Epidemiology and Psychopathology, Site de Cery, 1008 Prilly, Switzerland. E-mail: sirak.gebreab@chuv.ch

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over lifetime. Besides, the effect of stress during childhood and adulthood on the risk of adult cardiometabolic abnormality has been studied separately most of the time, which has impeded drawing conclusions regarding the independent effect stress within these distinct periods. In line with the stress sensitization hypothesis (22,23), previous stress may amplify the risk of cardiometabolic abnormality in case of new exposure to life events. Life course epidemiology also depicts physical and/or social exposures at different periods in life have biological, behavioral, and psychosocial pathways with lasting effects on body systems and implications for disease (24,25). Indeed, personality traits and coping mechanisms have been shown to act as risk and resilience factors for the effect of stress (26–30). Studies have also revealed associations between psychosocial stress and major depressive disorder (MDD) (31) and between MDD and CVDs (32). Recent research suggests that the association between MDD and cardiometabolic risk factors is entirely attributable to the atypical depression subtype (characterized by increased appetite, increased sleep, and leaden paralysis) (33–36). However, the interplay between psychosocial stress and psychological factors on the risk cardiometabolic abnormality has hardly been studied.

Accordingly, to better understand the link between lifetime psychosocial stress and cardiometabolic risk, (1) we independently assessed the associations between childhood adversity, life-event stress in remote and recent adulthood, and cardiometabolic risk indicators including obesity markers, blood glucose and lipid levels, blood pressure, and a composite cardiometabolic risk score in a population-based sample; (2) we tested whether exposure to childhood adversity and adulthood life-event stress interact to affect the cardiometabolic risk measures; (3) we determined whether psychological factors such as personality traits, coping styles, and MDD subtypes accounted for the associations between psychosocial stress and the cardiometabolic risk measures or modified their effects.

METHODS

Study Sample and Design

Data for the present study came from the ongoing population-based prospective cohort study CoLaus|PsyCoLaus, which was designed to study cardiovascular risk factors and mental disorders in the community (37,38). A total of 6733 randomly selected individuals, based on the civil registry, aged between 35 and 75 years, were recruited between June 2003 and May 2006 in the city of Lausanne (Switzerland). Sixty-seven percent of the 35- to 66-year-old participants who underwent the physical examination (n = 5535) also accepted the psychiatric evaluation (n = 3719), which took place from January 2004 to May 2009. For the present analyses, 2674 participants with complete information on psychosocial stress and cardiometabolic risk indicators as well as on personality traits, coping strategies, and MDD could be included (Fig. 1). Participants who could not be included were more likely to be men, to be nonwhite, to have a lower educational level, and to be physically inactive. The institutional ethics committee of the University of Lausanne approved the CoLaus |PsyCoLaus study. All participants signed a written informed consent after having received a detailed description of the study.

Assessment

Cardiometabolic Measurements

The physical evaluation included anthropometric measurements and the collection of blood and urine samples (37). Body weight and height were measured, and the body mass index (BMI) was calculated. Waist circumference was measured at the narrowest point between the lowest rib and the iliac crest. Body fat mass was assessed using bioelectrical impedance analysis. Blood pressure was measured three times on the left arm with 10-minute intervals; the average of the last two measures was then used. Venous blood samples (50 ml) were drawn after an overnight fast and clinical chemistry assay was performed within 2 hours of blood collection. High density lipoprotein (HDL) was assessed by cholesterol oxidase-phenol-aminophenazone + polyethylene glycol + cyclodextrin with a maximum interassay CV of 3.6% and a maximum intra-assay CV of 0.9%. Triglycerides were assessed by glucose oxidase-phenol-aminophenazone with a maximum interassay CV of 2.9% and a maximum intra-assay CV of 1.5%. Apolipoprotein B quantification was performed by turbidimetry with a maximum interassay CV of 8.7% and a maximum intra-assay CV of 7.6%. Glucose was assessed by glucose dehydrogenase with a maximum interassay CV of 2.1% and a maximum intra-assay CV of 1.0%. A continuous cumulative cardiometabolic risk score was also created on the basis of the cardiometabolic risk indicators including, BMI, waist circumference, body fat mass, levels of triglycerides, HDL (inversed levels), apolipoprotein B, as well as glucose and systolic blood pressure (SBP). The continuous cardiometabolic risk score was the sum of the sex-specific standardized z-scores of the indicators where the participant had valid measurements in all indicators (39). As most of the indicators had skewed distributions (see Supplementary A, Supplemental Digital Content, http://links.lww.com/PSYMED/A496), appropriate power transformations were applied before calculating the scores (see Supplementary B, Supplemental Digital Content, http://links.lww.com/PSYMED/A496). The suitability of aggregating indicators in a composite measure was tested using confirmatory factor analysis (see Supplementary C, Supplemental Digital Content, http://links.lww.com/PSYMED/A496) which revealed an acceptable fit for a common factor solution (e.g., root mean square error of approximation = 0.03, comparative fit index = 0.98, normed fit index = 0.97).

Psychosocial Stress

Information about early-life adversity was elicited using questions on childhood events from the Schedule for Affective Disorders and Schizophrenia-Lifetime Version (40). The following stressful experiences until the age of 16 were considered as indicators of adversity: loss of close relatives (parents or siblings), divorce or separation of parents, witnessing of violence between parents, as well as sexual and physical abuse (41). According to the suggestion of Friedman et al. (14), exposure to childhood adversity was quantified by the sum of reported events. Stressful life events during adulthood were evaluated using the Amiel-Lebigre's Life Event Questionnaire (42). The questionnaire comprises a list of 52 events related to the family environment, relationships, health issues, the social environment, work and employment, the legal system, housing, and economic problems (Table 1). When participants reported that they had been exposed to a specific adverse event, they were asked to provide the age of the first exposure to this event. If the event had occurred more than once, they also provided the age of the last exposure to this event. In addition, they were asked to rate the negative affective impact of the first and the last occurrences of the event by a score ranging from 0 (no negative impact at all) to 100 (maximal negative impact the participant could imagine). The questionnaire was previously evaluated by its originators in samples of depressed individuals and controls (42). The cumulative severity score of events of 200 or more for the 2 preceding years was found to indicate the presence of depression (43). Among depressive women and men, 56% and 74%, respectively, had a score of 200 and more, whereas in controls, the respective proportions for these scores were only 30% and 12%. Given that participants provided us with the timing of each event, we could compute cumulative severity scores for the events reported for the 5 years preceding the interview (recent adult life-event stress) and for the period earlier than the 5 years preceding the interview (remote adult life-event stress). The 5-year cut-off was chosen according to the predictive power of the cumulative severity scores of

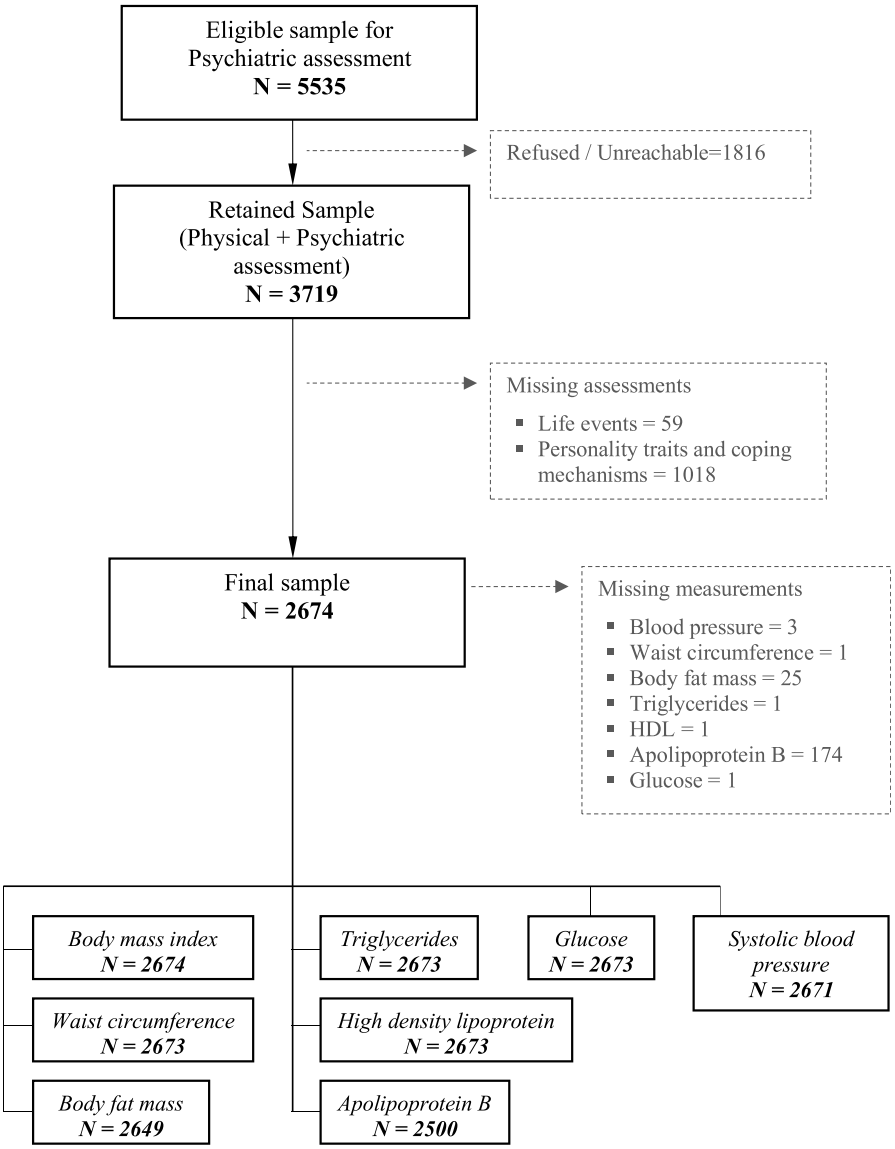

FIGURE 1. Sample flow chart.

stressors on the total score of the General Health Questionnaire (44,45). By varying the duration of the exposure to stressors from the most recent 1-year period up to a maximum of 10 years, we could establish that the prediction of the General Health Questionnaire score did not further improve in terms of the area under the curve when an exposure period of longer than 5 years was considered. This cut-off was also confirmed using the Akaike Information Criterion within a linear model framework (see Supplementary Table D, Supplemental Digital Content, http://links.lww.com/PSYMED/A496). To increase the interpretability, we rescaled the scores to range from 0 to 100. Accordingly, one-unit increase corresponds to a 1% increase of the maximum score in the sample.

Psychological Factors

MDD was assessed using the French version of the semistructured Diagnostic Interview for Genetic Studies (46,47), which revealed excellent interrater ($\kappa = 0.93$) and adequate 6-week test-retest reliability ($\kappa = 0.62$) for MDD, tested in a clinical sample (46). Interviewers were required to be master-level psychologists, who were trained for 1 to 2 months. The Diagnostic Interview for Genetic Studies information also elicits the criteria for the atypical and melancholic characteristics of major depressive episodes. According to the lifetime history of these episodes, subtypes with MDD were classified into the atypical, melancholic, combined, and unspecified subtypes as suggested by Angst et al. (34,48,49). The personality dimensions of Neuroticism and Extraversion were evaluated using the Eysenck Personality Questionnaire (50).The originator of this instrument reported Cronbach's α coefficients of 0.78 to 0.87 for neuroticism and 0.72 to 0.82 for extraversion using three different samples in France (51). Coping strategies were evaluated using the coping section of the problem resolution strategy questionnaire (52). According to principal component analysis, factors emotion-focused coping, help-seeking behaviors, and problem-focused coping were identified. Using the CoLaus|PsyCoLaus baseline sample, we established standardized Cronbach's α coefficients for these dimensions of 0.65, 0.69, and 0.44, respectively (53). As emotion-focused coping was highly correlated with neuroticism ($r = 0.63$, $p < .0001$), we could not include it in our analyses.

TABLE 1. Stressful Life Events

Events related to family environment
Alcoholism in a close family member
Suicide of a close family member
Death of a close family member
Severe accident or disease within the close family
Arrival of a new family member in the home
Pregnancy
Increased arguments with 1 or more members of close family
Problem with family other than close family members (e.g., in-laws)
Child leaving home
Necessity to have others look after children
Behavioral problems with children
Events with spouse or partner
Unwanted pregnancy by one of the partners
Sexual problems within the couple
Marriage
Increased arguments with spouse or partner
Death of partner
Divorce or separation from partner
Separation from partner imposed by circumstances
Extramarital/extrapartner affair
Breakup of relationship
Infidelity of partner
Reconciliation with partner
Events related to health/psychological problems
Disease or severe injury requiring hospitalization
Disease or accident requiring medical treatment
Sudden severe hearing or visual handicap
Miscarriage
Abortion
Problems related to alcohol and drug
Events related to social environment and other psychosocial problems
Retirement
Moving
Change of neighbors
Quarrel with neighbors
Social life diminution
Death of a close friend
Events related to work and employment
Unemployment (a participant)
Unemployment (a partner)
Problems with boss or colleagues
Job change of the same type
Job change of different type
Change of work hours or conditions
Promotion or increased responsibility at work
Professional failure
Partner starting or stopping his/her job
Events related to housing/economic problems
Significant income increment
Significant income decrement
Becoming highly indebted
Small financial difficulties
Loss of object with high personal value
Homelessness for a certain time
Events related to the legal system
Sentenced to imprisonment
Participation in a fight
Imprisonment

Continued in next column

Sociodemographics, Lifestyle Factors, and Other Covariates

Data on sociodemographic characteristics (sex, age, education, and income), health-related lifestyle factors (alcohol consumption, smoking status, and physical inactivity), cardiometabolic abnormalities in first-degree relatives (diabetes, hypertension, or hypercholesterolemia), and the use of physician-prescribed medication (antidiabetic, antihypertensive, lipid-lowering, and other weight-increasing medications) were collected through a standardized interview. Education was categorized into the following five levels: (1) compulsory school (maximum 10 school years); (2) apprenticeship; (3) upper secondary education (maximum 13 school years); (4) higher education except for university; and (5) university. Yearly income was assessed in Swiss Francs in the following six categories: (1) <30,000; (2) 30,000 to 49,000; (3) 50,000 to 69,000; (4) 70,000 to 89,000; (5) 90,000 to 110,000; and (6) greater than 110,000. Alcohol consumption was determined through the type and number of alcoholic beverage units consumed on a weekly basis (54). Self-reported tobacco consumption was used to determine smoking status. Participants were classified as nonsmokers if they had never regularly smoked, as former smokers if they had a history of smoking in past but had stopped smoking and as current smokers if they reported a current regular consumption. Participants were considered to be physically active if they reported to perform physical activity for at least 20 minutes twice a week.

Statistical Analysis

First, marginal associations between childhood adversity, adulthood remote and recent stress, and cardiometabolic variables (BMI, waist circumference, body fat mass, triglycerides, HDL, apolipoprotein B, fasting blood glucose, SBP, and the cumulative cardiometabolic risk score) were established using robust linear regression models (M-estimation). Then, the conditional effects of childhood adversity and both remote and recent adulthood stress on these cardiometabolic indicators were assessed within models with adjustment for sociodemographic characteristics, family history of cardiometabolic risk, lifestyle factors, and the use of medication (Model 1); we also tested whether the effects of childhood and adulthood stress interacted regarding the cardiometabolic risk indicators. In the next series of models, psychological factors including personality traits, coping style, and MDD subtypes were entered (Model 2). Potential effect modification by psychological factors was tested by adding interaction terms of these psychological factors and childhood adversity or adulthood stress to the models. The percentage of change in the effects of stress variables after including a group of potential confounding variables or potential mediators in the model was calculated using the formula: $\Delta = \left(\frac{\beta \text{ Model with adjustments} - \beta \text{ Reference model}}{\beta \text{ Model with adjustments}}\right) \times 100\%$.

In all analyses, observations were weighted by their inverse probability of being in the sample. This inverse probability was derived from a model that included sociodemographic (age, sex, educational level, income level,

race) and lifestyle (smoking status, physical activity, alcohol consumption) predictors of participation. Missing information on the covariates was attributed by multiple imputations (n = 100, Markov Chain Monte Carlo method) under the missing-at-random assumption; observations with missing information in the outcome variables were dropped from the analysis. Statistical significance throughout the analyses was maintained at the 0.05 alpha levels and a 0.01 alpha level was used to test interactions. Analyses were carried out using the Statistical Analysis System (SAS), Version 9.3 (SAS institute Inc, Cary, NC) and R (http://www.cran.r-project.org).

RESULTS

Table 2 provides the description of the sample. Nearly half of the participants in our sample were overweight (BMI ≥ 25). The median score for the cumulative cardiometabolic risk was −0.29 ranging from −16.3 to 23.5, where 620 participants had a score above 3, representing the upper risk quartile. Nearly a third of the participants (31.2%) reported at least one adverse event during childhood. Among them, a quarter was exposed to two or more events. The most frequently reported event was witnessing of violence between parents (13.8%), followed by divorce or separation of parents (12.9%), death of a parent (8.0%), death of a sibling (2.5%), sexual abuse (2.4%), and physical abuse (0.8%). Half of the sample had also experienced up to 11 life events in their adulthood; only 3% reported not having experienced any event. Most of the reported events were described to have happened between the ages of 25 and 45 years. As many as 42.8% met lifetime criteria for MDD, of which half of them met criteria for the unspecified subtype.

Results of the multiple covariate regression models did not provide evidence for interactions between childhood adversity and remote or recent adulthood and between remote and recent adult stress regarding the cardiometabolic risk outcomes. Similarly, there were no significant interactions between psychological factors and childhood adversity or adulthood stress. However, these models revealed that participants exposed to childhood adversity had significantly increased BMI, waist circumference, and body fat (Table 3, model 1) as well as increased SBP levels (Table 4, Model 1) and cumulative cardiometabolic risk score (Table 5, Model 1). These associations also remained significant after adjustment for psychological variables, which hardly diminished the established effect sizes. The association between childhood adversity and decreased levels of HDL only reached statistical significance after adjustment for the psychological variables (Table 4, Model 2). Regarding adulthood stress, remote life-event stress was positively associated with BMI, waist circumference (Table 3, Model 1), apolipoprotein B level (Table 4, Model 1), and the cumulative cardiometabolic risk score (Table 5, Model 1). The adjustment for psychological factors had a very limited effect on the measured effect sizes for these associations. In contrast, recent adulthood stress was associated with lower waist circumference, lower apolipoprotein B level, and lower systolic blood pressure with and without adjustment for psychological characteristics (Table 4).

DISCUSSION

Using a population-based study, which relied on thorough physical and psychiatric evaluations, the key findings of the present analyses were the following: (1) childhood adversity and adult life-event stress were independently associated with increased levels of obesity markers, abnormal blood lipids, and a higher cumulative cardiometabolic risk score; (2) only life-event stress in remote but not in recent adulthood was associated with elevated levels of these outcomes; and (3) personality traits, coping strategies, or MDD subtypes did not account for the associations between stress and cardiometabolic risk and these psychological factors were not modifiers of these associations.

Stress During Childhood and Cardiometabolic Risk

Exposure to one more adverse childhood event was significantly associated with elevated BMI, waist circumference, fat mass, SBP, cardiometabolic risk score, and low HDL levels after adjustment for sociodemographic, lifestyle, and psychological factors, which is particularly remarkable given the advanced age of our sample. Our findings are in line with growing evidence showing associations between stress during early life and the risk of cardiometabolic abnormality (14,21). A review by Danese and Tan (17), for example, has summarized findings on childhood maltreatment and obesity and concluded that childhood adversity is a potential risk factor for abnormal weight. A study by Lee et al. (55) has shown that individuals with a history of abuse during childhood (emotional, physical, and sexual) have a greater risk of developing the metabolic syndrome. Similarly, Winning et al. (21) found that psychological distress in childhood was associated with a higher cardiometabolic risk later on in life. A 20-year follow-up study has also revealed that participants who were exposed to multiple adverse childhood experiences had greater increase of SBP levels in young adulthood (56). In addition to these studies, we could show that the association between childhood adversity and cardiometabolic risk was independent of later life stress, personality traits, coping mechanisms, and the occurrence of a MDD.

It has often been hypothesized that stress during childhood affects cardiometabolic functioning indirectly by precipitating health-related behaviors such as sedentary lifestyle, poor diet, smoking, and heavy alcohol use that may persist all through adulthood (16,57). As far as lifestyle factors were assessed in our study, our results did not support their mediating role. However, lifestyle factors can change over the lifespan and earlier lifestyle factors may have been more relevant as mediators of the effect of adverse childhood events on cardiometabolic outcome variables than the lifestyle factors that we assessed at an already more advanced age. Moreover, we were lacking data on diet and only had information on regular physical exercise at the time of the interview rather than on overall physical activity. Nevertheless, the fact that childhood adversity remained significantly associated with cardiometabolic variables after accounting for such health-related lifestyle suggests other pathways that could be related to the biological system (58). Childhood is a sensitive period where exposures can have adverse effects on structures and functioning of the body system (59). Continuous exposure to stressors during this period may affect the biological stress response and/or produce epigenetic changes (58), which could lead to cardiometabolic abnormalities later on in life. These stress-related biological processes and their links to the subsequent cardiometabolic risk need to be elucidated in future research.

Stress During Adulthood and Cardiometabolic Risk

Regarding stress during adulthood, we found remote rather than recent life-event stress to be a risk factor for elevated levels of cardiometabolic risk indicators. The differential effect of stress

TABLE 2. Sample Characteristics (n = 2674)

Sociodemographics	
Females, *n* (%)	1479 (55.3%)
Age, M (SD)	50.4 (8.8)
Income level, *n* (%)	
<30,000 CHF	152 (5.8%)
30,000–49,999 CHF	393 (15%)
50,000–69,999 CHF	584 (22.3%)
70,000–80,999 CHF	513 (19.6%)
90,000–110,000 CHF	386 (17.7%)
>110,000 CHF	598 (22.6%)
Educational level, *n* (%)	
Compulsory school	411 (15.3%)
Apprenticeship	1005 (37.5%)
Upper secondary school	245 (9.2%)
Higher education except for university	427 (16%)
University	586 (22%)
Family history of cardiometabolic risk,[a] *n*(%)	1321 (49.4%)
Health-related lifestyle	
Smoking status, *n* (%)	
Nonsmoker	1073 (40.1%)
Former smoker	717 (26.8%)
Current smoker	884 (33.1%)
Physically inactive (<20 min twice a week), *n* (%)	1119 (41.9%)
No. alcoholic drinks per week, median (range)	4 (0–76)
Cardiometabolic risk indicators	
BMI, M (SD), kg/m^2	25.4 (4.5)
Waist circumference, M (SD), cm	87.6 (13.3)
Body fat mass, M (SD), %	28.5 (8.7)
Triglycerides, M (SD), mmol/l	1.3 (1.1)
HDL, M (SD), mmol/l	1.6 (0.4)
Apolipoprotein B, M (SD), mg/dl	166 (122.6)
Fasting blood glucose, M (SD), mmol/l	5.5 (1.1)
Systolic blood pressure, M (SD), mm Hg	125.4 (16.5)
Cumulative cardiometabolic risk score,[b] M (SD)	0.006 (4.8)
Psychosocial stress	
Adulthood life-event stress,[c] median (range)	310 (0–2520)
Recent (<5 y) life-event stress,[c] median (range)	70 (0–825)
Remote (>5 y) life-event stress,[c] median (range)	215 (0–2250)
Childhood adverse events, *n* (%)	
No event	1242 (68.8%)
1 event	1047 (23.5%)
2 events	301 (6.4%)
≥3 events	84 (1.3%)
Psychological factors	
Neuroticism score, M (SD)	9.7 (5.8)
Extraversion score, M (SD)	11.9 (5.0)
Problem-focused coping score, M (SD)	7.7 (1.8)
Help-seeking coping score, M (SD)	4.3 (2.6)
Major depressive disorder subtypes	
Atypical, *n* (%)	184 (6.9%)
Melancholic, *n* (%)	287 (10.7%)
Combined, *n* (%)	137 (5.1%)
Unspecified, *n* (%)	538 (20.1%)

M = mean; SD = standard deviation; CHF = Swiss franc; BMI = body mass index; HDL = high density lipoprotein.

[a] History of either diabetes, hypertension, or hypercholesterolemia in first-degree relatives.

[b] The sum of sex-specific standardized z-scores of BMI, waist circumference, body fat mass, triglycerides, HDL, apolipoprotein B, fasting blood glucose, and systolic blood pressure.

[c] Cumulative severity scores of life events.

in function of the 5-year dichotomy is likely to be explained by the duration/exposure window. Indeed, there may be an interval of several years between the occurrence of stress and the manifestation of significant weight increase or before developing other cardiometabolic risk factors. According to our data, the short-term effect of stress may be rather the converse. Indeed, increased stress during the 5 years before the assessment was associated with decreasing waist circumference, apolipoprotein B levels, and SBP. The observed decrease in waist circumference suggests that the negative associations between stress and apolipoprotein B concentrations and SBP could be attributable to decreased appetite and subsequent weight loss in stressful periods (60,61). Our finding of negative associations between recent stress and three cardiometabolic outcome variables are in line with a Swedish study that also found negative correlations between the number of negative life events experienced during the year preceding the assessment and waist circumference, blood pressure, and lipid levels in white-collar workers (62). In contrast to the results of this Swedish study and ours, reviews and meta-analyses have rather revealed a tendency for positive associations between stress and obesity markers (4), dyslipidemia, and hypertension (4,11). However, studies included in the review and the meta-analysis that predominantly focused on chronic stressors hardly distinguished between recent and remote life stress.

In contrast to recent stress, we found a positive association between remote stress and increased BMI and waist circumference, whereas the associations for the fat mass did not reach the threshold of statistical significance. Our finding of an association between stress and adiposity markers is consistent with those of several previous studies (4–6). Interestingly, we also found a positive association between remote life-event stress and apolipoprotein B blood concentrations, an indicator of the number of circulating atherogenic lipid particles in the blood. No study has previously assessed the association between psychosocial stress and apolipoprotein B, although recent research suggested that higher apolipoprotein B levels could be the most important predictor of cardiovascular events (63–65). Accordingly, our finding of a positive association between life-event stress and apolipoprotein B concentrations provides additional evidence for a potential pathway from stress to cardiovascular outcomes, which could be via abnormal lipid metabolism. We also found a significant association between remote life-event stress and the aggregated cardiometabolic risk measure. This finding highlights the considerable effect of life events in the

TABLE 3. Associations Between Psychosocial Stress and Obesity Markers in Bivariate and Multiple Covariate Regression Models

	Bivariate	Model 1		Model 2	
	β (95% CI)	β (95% CI)	Δ	β (95% CI)	Δ
BMI, kg/m^2 (n = 2674)					
Recent (<5 y) life-event stress	−0.018* (−0.033 to −0.004)	−0.009 (−0.022 to 0.005)		−0.012 (−0.025 to 0.002)	
Remote (>5 y) life-event stress	0.012 (−0.002 to 0.026)	0.019** (0.006 to 0.033)	19%	0.020** (0.006 to 0.034)	5%
Childhood adversity	0.262* (0.063 to 0.462)	0.276* (0.056 to 0.495)	−5%	0.249* (0.029 to 0.468)	−5%
Neuroticism	−0.149 (−0.310 to 0.011)			−0.049 (−0.219 to 0.121)	
Extraversion	0.164* (0.001 to 0.326)			0.290*** (0.136 to 0.443)	
Problem-focused coping	0.017 (−0.143 to 0.177)			0.012 (−0.139 to 0.163)	
Help-seeking coping	−0.353*** (−0.512 to −0.194)			−0.032 (−0.187 to 0.123)	
Atypical depression	0.715* (0.041 to 1.390)			1.355*** (0.715 to 1.994)	
Melancholic depression	−1.024*** (−1.527 to −0.521)			−0.335 (−0.846 to 0.175)	
Combined depression	−0.236 (−1.046 to 0.574)			0.038 (−0.739 to 0.815)	
Unspecified depression	−0.518* (−0.915 to −0.120)			−0.149 (−0.540 to 0.242)	
Waist circumference, cm (n = 2673)					
Recent (<5 y) life-event stress	−0.123*** (−0.173 to −0.073)	−0.039* (−0.076 to −0.001)	−209%	−0.044* (−0.082 to −0.007)	11%
Remote (>5 y) life-event stress	0.017 (−0.031 to 0.065)	0.060** (0.023 to 0.097)	90%	0.061** (0.024 to 0.099)	1%
Childhood adversity	0.264 (−0.439 to 0.966)	0.648* (0.044 to 1.253)	40%	0.608* (0.001 to 1.215)	−6%
Neuroticism	−1.038*** (−1.595 to −0.481)			−0.213 (−0.684 to 0.258)	
Extraversion	0.362 (−0.203 to 0.928)			0.541* (0.117 to 0.966)	
Problem-focused coping	0.189 (−0.372 to 0.751)			−0.058 (−0.474 to 0.358)	
Help-seeking coping	−1.726*** (−2.278 to −1.174)			0.048 (−0.380 to 0.477)	
Atypical depression	0.850 (−1.531 to 3.230)			3.153*** (1.383 to 4.922)	
Melancholic depression	−4.410*** (−6.149 to -2.670)			−0.554 (−1.966 to 0.858)	
Combined depression	−1.786 (−4.667 to 1.094)			0.395 (−1.754 to 2.544)	
Unspecified depression	−2.709*** (−4.092 to −1.326)			−0.531 (−1.615 to 0.553)	
Body fat mass, % (n = 2649)					
Recent (<5 y) life-event stress	0.010 (−0.024 to 0.043)	−0.004 (−0.025 to 0.017)	340%	−0.008 (−0.029 to 0.013)	33%
Remote (>5 y) life-event stress	0.160*** (0.129 to 0.191)	0.020 (−0.001 to 0.040)		0.016 (−0.006 to 0.037)	−6%
Childhood adversity	1.076*** (0.611 to 1.542)	0.555** (0.215 to 0.896)	−95%	0.506** (0.165 to 0.846)	−4%
Neuroticism	0.947*** (0.575 to 1.319)			0.023 (−0.240 to 0.286)	
Extraversion	0.068 (−0.309 to 0.445)			0.350** (0.112 to 0.589)	
Problem-focused coping	0.236 (−0.134 to 0.605)			0.264* (0.030 to 0.499)	
Help-seeking coping	0.545** (0.174 to 0.915)			0.197 (−0.043 to 0.438)	
Atypical depression	4.345*** (2.807 to 5.882)			2.036*** (1.046 to 3.026)	
Melancholic depression	1.278* (0.098 to 2.457)			−0.302 (−1.095 to 0.491)	
Combined depression	4.429*** (2.572 to 6.285)			1.000 (−0.200 to 2.199)	
Unspecified depression	1.142* (0.215 to 2.069)			0.055 (−0.552 to 0.663)	

CI = confidence interval; BMI = body mass index.

Model 1: model with psychosocial stress variables adjusted for sociodemographics (sex, age, income level, educational level), family history of cardiometabolic risk, health-related lifestyle factors (alcohol consumption, smoking status, physical inactivity), and the use of weight-increasing medication.

Model 2: model 1 with psychological factors (personality traits, coping strategies, and major depressive disorder subtypes).

Betas associated with recent and remote stress are a 1% increase in the range of that variable.

Betas associated with neuroticism, extraversion, problem-focused coping, help-seeking coping are 1 SD increase for that variable.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

$$\Delta = \left(\frac{\beta \text{ Model with adjustments} - \beta \text{ Reference model}}{\beta \text{ Model with adjustments}}\right) \times 100\%$$

overall cardiometabolic functioning, which, in consequence, may culminate in cardiovascular and metabolic disease. The significant associations with the cumulative risk measure and the other cardiometabolic risk indicators were all independent of sociodemographic, lifestyle factors, and psychological factors. However, the relationship between stress, the socioeconomic factors income and

TABLE 4. Associations Between Psychosocial Stress Lipids, Blood Glucose, and Systolic Blood Pressure in Bivariate and Multiple Covariate Regression Models

	Bivariate	Model 1		Model 2	
	β (95% CI)	β (95% CI)	Δ	β (95% CI)	Δ
Triglycerides, mmol/l (n = 2673)					
Recent (<5 y) life-event stress	−0.001 (−0.003 to 0.001)	0.001 (−0.002 to 0.003)		0.000 (−0.002 to 0.003)	
Remote (>5 y) life-event stress	0.001 (−0.001 to 0.003)	0.000 (−0.002 to 0.002)		0.000 (−0.002 to 0.002)	
Childhood adversity	−0.011 (−0.035 to 0.013)	0.000 (−0.034 to 0.033)		−0.001 (−0.034 to 0.033)	
Neuroticism	−0.019* (−0.039 to 0.000)			−0.007 (−0.033 to 0.019)	
Extraversion	0.011 (−0.008 to 0.031)			0.042*** (0.019 to 0.065)	
Problem-focused coping	−0.018 (−0.037 to 0.001)			−0.003 (−0.026 to 0.020)	
Help-seeking coping	−0.029** (−0.048 to −0.009)			0.010 (−0.014 to 0.033)	
Atypical depression	0.032 (−0.048 to 0.113)			0.040 (−0.058 to 0.137)	
Melancholic depression	−0.047 (−0.108 to 0.014)			0.009 (−0.069 to 0.086)	
Combined depression	−0.132** (−0.229 to −0.035)			−0.036 (−0.154 to 0.082)	
Unspecified depression	−0.028 (−0.075 to 0.020)			0.015 (−0.044 to 0.075)	
HDL, mmol/l (n = 2673)					
Recent (<5 y) life-event stress	−0.001 (−0.002 to 0.001)	−0.001 (−0.002 to 0.000)		−0.001 (−0.002 to 0.000)	
Remote (>5 y) life-event stress	0.000 (−0.001 to 0.002)	−0.001 (−0.002 to 0.001)		−0.001 (−0.002 to 0.001)	
Childhood adversity	−0.014 (−0.037 to 0.009)	−0.020 (−0.041 to 0.001)		−0.021* (−0.042 to 0.000)	***6%***
Neuroticism	0.012 (−0.006 to 0.030)			−0.001 (−0.017 to 0.016)	
Extraversion	−0.016 (−0.034 to 0.003)			−0.015 (−0.029 to 0.000)	
Problem-focused coping	−0.009 (−0.028 to 0.009)			0.010 (−0.004 to 0.025)	
Help-seeking coping	0.025** (0.008 to 0.043)			−0.007 (−0.022 to 0.008)	
Atypical depression	0.052 (−0.027 to 0.130)			0.054 (−0.008 to 0.115)	
Melancholic depression	0.090** (0.032 to 0.148)			−0.003 (−0.052 to 0.046)	
Combined depression	0.076 (−0.019 to 0.170)			−0.004 (−0.078 to 0.071)	
Unspecified depression	0.021 (−0.025 to 0.068)			0.015 (−0.023 to 0.052)	
Apolipoprotein B, mg/dl (n = 2500)					
Recent (<5 y) life-event stress	−0.344** (−0.580 to −0.109)	−0.443** (−0.728 to −0.157)		−0.437** (−0.724 to −0.150)	
Remote (>5 y) life-event stress	0.265* (0.034 to 0.496)	0.626*** (0.338 to 0.913)		0.607*** (0.312 to 0.901)	
Childhood adversity	−1.924 (−5.969 to 2.121)	−0.504 (−5.273 to 4.265)		−0.125 (−4.918 to 4.668)	
Neuroticism	−1.730 (−4.390 to 0.931)			−1.853 (−5.446 to 1.740)	
Extraversion	−0.940 (−3.620 to 1.740)			0.303 (−2.956 to 3.562)	
Problem-focused coping	−2.085 (−4.741 to 0.572)			−1.984 (−5.167 to 1.200)	
Help-seeking coping	−3.609** (−6.296 to −0.923)			−0.453 (−3.819 to 2.912)	
Atypical depression	−8.415 (−19.761 to 2.932)			−2.016 (−15.903 to 11.871)	
Melancholic depression	−4.370 (−12.888 to 4.148)			−0.737 (−11.751 to 10.277)	
Combined depression	2.582 (−10.913 to 16.078)			22.707** (6.227 to 39.188)	
Unspecified depression	4.997 (−1.628 to 11.622)			10.938* (2.586 to 19.290)	
Fasting blood glucose, mmol/l (n = 2673)					
Recent (<5 y) life-event stress	−0.002* (−0.004 to 0.000)	−0.001 (−0.003 to 0.001)		−0.001 (−0.003 to 0.001)	
Remote (>5 y) life-event stress	0.000 (−0.002 to 0.002)	0.001 (−0.001 to 0.003)		0.001 (−0.001 to 0.003)	
Childhood adversity	0.006 (−0.021 to 0.032)	0.012 (−0.019 to 0.044)		0.012 (−0.020 to 0.043)	
Neuroticism	−0.034** (−0.055 to −0.012)			−0.001 (−0.026 to 0.023)	
Extraversion	0.000 (−0.021 to 0.022)			0.012 (−0.010 to 0.034)	
Problem-focused coping	−0.008 (−0.029 to 0.013)			−0.010 (−0.032 to 0.011)	
Help-seeking coping	−0.050*** (−0.071 to −0.029)			−0.005 (−0.027 to 0.017)	
Atypical depression	−0.069 (−0.158 to 0.020)			0.018 (−0.073 to 0.110)	
Melancholic depression	−0.106** (−0.173 to −0.038)			−0.028 (−0.102 to 0.045)	

Continued on next page

TABLE 4. *(Continued)*

	Bivariate	Model 1		Model 2	
	β (95% CI)	β (95% CI)	Δ	β (95% CI)	Δ
Combined depression	−0.060 (−0.167 to 0.048)			0.027 (−0.085 to 0.138)	
Unspecified depression	−0.087** (−0.140 to −0.034)			−0.071* (−0.128 to −0.015)	
Systolic blood pressure, mm Hg (n = 2671)					
Recent (<5 y) life-event stress	−0.151*** (−0.209 to −0.092)	−0.059* (−0.107 to −0.011)		−0.059* (−0.107 to −0.010)	
Remote (>5 y) life-event stress	−0.002 (−0.057 to 0.054)	−0.035 (−0.083 to 0.013)		−0.030 (−0.079 to 0.019)	
Childhood adversity	0.025 (−0.786 to 0.835)	0.974* (0.191 to 1.757)		0.952* (0.165 to 1.740)	
Neuroticism	−1.575*** (−2.221 to −0.929)			−0.577 (−1.185 to 0.031)	
Extraversion	−0.222 (−0.877 to 0.433)			−0.065 (−0.617 to 0.488)	
Problem-focused coping	0.554 (−0.093 to 1.201)			0.610* (0.071 to 1.149)	
Help-seeking coping	−1.899*** (−2.536 to −1.262)			−0.159 (−0.713 to 0.395)	
Atypical depression	−1.058 (−3.773 to 1.658)			1.233 (−1.063 to 3.530)	
Melancholic depression	−3.373** (−5.428 to −1.317)			−0.386 (−2.221 to 1.449)	
Combined depression	−2.774 (−6.056 to 0.508)			0.098 (−2.690 to 2.885)	
Unspecified depression	−2.291** (−3.899 to −0.683)			−0.270 (−1.675 to 1.135)	

CI = confidence interval.

Model 1: model with psychosocial stress variables adjusted for sociodemographics (sex, age, income level, educational level), family history of cardiometabolic risk, health-related lifestyle factors (alcohol consumption, smoking status, physical inactivity), and the use of weight-increasing medication.

Model 2: model 1 with psychological factors (personality traits, coping strategies, and major depressive disorder subtypes).

Betas associated with recent and remote stress are a 1% increase in the range of that variable.

Betas associated with neuroticism, extraversion, problem-focused coping, and help-seeking coping are 1 SD increase for that variable.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

$$\Delta = \left(\frac{\beta \text{ Model with adjustments} - \beta \text{ Reference model}}{\beta \text{ Model with adjustments}}\right) \times 100\%$$

education, and cardiometabolic risk is complex, and there is overlap, particularly between events related to unemployment or economic problems and income. Accordingly, adjustment for socioeconomic variables is likely to provide a conservative estimate of the effect of stress (66).

Lifetime Psychosocial Stress and Cardiometabolic Risk

According to the stress sensitization hypothesis, early-life stress increases responsiveness to subsequent stressors by imposing biological alterations (23,24), whereas other hypotheses posit that stress in early life and later periods has a cumulative effect on cardiovascular risk (16,67). Our observation of independent effects of childhood adversity and adulthood stress on cardiometabolic risk indicators, together with the absence of an interaction between childhood and adulthood stress regarding the cardiometabolic outcomes, provides support for additive effects of life stressors rather than for the stress sensitization hypothesis. Moreover, our data revealed that personality traits, coping style, and MDD subtypes did not change the magnitude of the established associations between stress and cardiometabolic risk outcomes, suggesting that these psychological characteristics are hardly mediators of the observed life course associations. The lifetime prevalence of more than 40% for MDD was high in our sample. As discussed in a recent article (68), this high prevalence was likely to be attributable to our recruitment in an urban area and the use of a semistructured diagnostic interview conducted by trained psychologist rather than a fully structured interview conducted by lay interviewers. In addition, our semistructured interview used a low threshold in the screening questions to enter the depression section to also assess disorders below the diagnostic threshold of MDD. However, considering that we found significant associations between MDD subtypes and cardiometabolic variables in our analyses, the absence of an effect of MDD subtypes on the associations between life-event stress and cardiometabolic variables is hardly attributable to the high prevalence of MDD. Hence, the absence of an effect of MDD subtypes on these associations is rather explained by the absence of an association between life-event stress and the atypical subtype (36), whereas this subtype has repetitively been shown to be associated with cardiovascular risk factors (33–36). Conversely, the unspecified MDD subtype is likely to be strongly associated with life-event stress (36), but not with cardiovascular risk (34,35,49).

STRENGTH AND LIMITATIONS

The strengths of our analyses were the assessment of stressful events over lifetime as well as the personal evaluation of the stressfulness of adult life events. Indeed, the bulk of previous studies have used the number of stressful events as a proxy measure of stress exposure. However, this approach does not take into account interindividual differences in the perception of the stressfulness of

TABLE 5. Associations Between Psychosocial Stress and the Cumulative Cardiometabolic Risk Score in Bivariate and Multiple Covariate Regression Models

	Bivariate	Model 1		Model 2	
	β (95% CI)	β (95% CI)	Δ	β (95% CI)	Δ
Cardiometabolic risk score† (n = 2478)					
Recent (<5 y) life-event stress	−0.030** (−0.049 to −0.011)	−0.011 (−0.026 to 0.005)		−0.012 (−0.027 to 0.004)	
Remote (>5 y) life-event stress	0.048*** (0.030 to 0.067)	0.016* (0.001 to 0.032)	−183%	0.017* (0.001 to 0.033)	7%
Childhood adversity	0.371** (0.103 to 0.639)	0.268* (0.007 to 0.528)	−28%	0.278* (0.017 to 0.540)	−4%
Neuroticism	−0.034 (−0.246 to 0.179)			−0.093 (−0.289 to 0.103)	
Extraversion	0.076 (−0.138 to 0.291)			0.250** (0.073 to 0.428)	
Problem-focused coping	−0.023 (−0.235 to 0.189)			−0.013 (−0.186 to 0.161)	
Help-seeking coping	−0.365*** (−0.579 to −0.151)			0.074 (−0.109 to 0.257)	
Atypical depression	0.575 (−0.327 to 1.477)			0.664 (−0.090 to 1.418)	
Melancholic depression	−0.903** (−1.584 to −0.222)			−0.372 (−0.973 to 0.229)	
Combined depression	0.567 (−0.499 to 1.633)			0.328 (−0.566 to 1.222)	
Unspecified depression	−0.290 (−0.824 to 0.244)			−0.010 (−0.465 to 0.445)	

CI = confidence interval.

Model 1: model with psychosocial stress variables adjusted for sociodemographics (sex, age, socioeconomic level), family history of cardiometabolic risk, health-related lifestyle factors (alcohol consumption, smoking status, physical inactivity), and the use of medication (antidiabetic, antihypertensive, lipid-lowering, and other weight-increasing medication).

Model 2: model 1 with psychological factors (personality traits, coping strategies, and major depressive disorder subtypes).

Betas associated with recent and remote stress are a 1% increase in the range of that variable.

Betas associated with neuroticism, extraversion, problem-focused coping, and help-seeking coping are 1 SD increase for that variable.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

$\Delta = \left(\frac{\beta \text{ Model with adjustments} - \beta \text{ Reference model}}{\beta \text{ Model with adjustments}}\right) \times 100\%$

† The sum of gender specific standardized z-scores of body mass index, waist circumference, body fat mass, triglycerides, high density lipoprotein (inversed levels), apolipoprotein B, fasting blood glucose and systolic blood pressure.

events. We also analyzed our data using the number of events as a measure of the exposure to adulthood stress rather than the scores that included perceived stressfulness. The results of these analyses (see Supplementary E, Supplemental Digital Content, http://links.lww.com/PSYMED/A496) generally revealed slightly lower associations with cardiometabolic outcome variables, which provide additional support to the use severity score. Indeed, adult life events were no longer associated with obesity markers and the cumulative cardiometabolic risk score. However, the associations with adipolipoprotein B and systolic blood pressure remained significant and significant associations appeared with the HDL. Our analyses also relied on a large population-based sample that was thoroughly evaluated with respect to cardiovascular risk factors, psychiatric disorders, and other psychological characteristics, which allowed us to evaluate the effect of life-event stress on cardiometabolic variables accounting for psychological factors.

However, the results of the study must also be viewed in the light of several limitations. First, data were assessed retrospectively and the recall of stressful events that partially occurred several decades before the assessment is likely to be incomplete and could have been biased by the participant's current state. Nonetheless, according to the recently published results of the population-based Dunedin cohort study, there is moderate agreement between retrospective and prospective measures of adverse childhood experiences, and both measures reveal associations with physical, mental, cognitive, and social health at midlife (69). Second, given the cross-sectional nature of data collection, it was not possible to determine the temporal sequence between life events and the onset of cardiometabolic abnormalities. Third, we had no data on diet and only self-reported information on regular physical exercise at the time of the interview rather than on overall physical activity, although both diet and physical activity could be potential mediators of the association between stress and cardiometabolic risk. Fourth, we had no data on the functioning of the hypothalamic-pituitary-adrenal axis and catecholamine. Indeed, chronic activation of the hypothalamic-pituitary-adrenal axis, triggered by repeated or continued exposure to stressful experiences for an extended period, could lead to a cascade of changes in the biological set-points that finally result in cardiometabolic changes (70). Fifth, data were collected in an urban sample in Switzerland, which may limit the generalizability of the findings.

CONCLUSIONS

Our findings provide additional insight into the complex relationship between life course psychosocial stress and cardiometabolic risk by showing that psychosocial stress in childhood as well as during adulthood is independently associated with adiposity and abnormal lipid profiles. The role of psychological factors in these associations seems to be trivial. Furthermore, stress encountered during these distinct periods may add up to affect overall cardiometabolic functioning, suggesting that individuals exposed to childhood adversity and high levels of stress during adulthood could particularly benefit from early screening of cardiovascular

risk factors. However, our findings still need to be confirmed by longitudinal research.

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